

Renal impairment in the elderly: a review¹

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Renal impairment is common in elderly patients. In one survey of acute admissions to a geriatric unit, 20% of all patients had quite significant renal impairment (Kafetz & Hodgkinson 1982). This problem is most often caused by the interaction of the fall-off in renal function which occurs with age and the propensity for dehydration amongst the elderly. There are differences in the approach to the assessment of renal impairment in older patients as well as in the nature of its causes. Renal failure in the elderly is usually prerenal in origin.

Fall-off in renal function with age

The age-related decline in renal function is one of the better known aspects of normal ageing, although its mechanism in both histological and physiological terms is not understood (McLachan 1978). Grossly, normal elderly kidneys at post-mortem look like kidneys of younger people with hypertension, so a diagnosis of hypertension should not be made at post-mortem in elderly patients on the grounds of renal changes alone. Perhaps a high pressure for a short period of time or a normal pressure for a longer period affect the kidney in the same way.

The mean glomerular filtration rate at the age of 30 is 140 ml/min/1.73m² and this falls to 97 ml/min/1.73m² at the age of 80 (Rowe *et al.* 1976a). Landahl and colleagues (1981) described a reference range of 48–100 ml/min/1.73m² for subjects aged between 70 and 75 years. The reduction in glomerular filtration rate with age is not linear but increases after the fifth decade.

It follows that the reference range for urea and creatinine is rather different in the elderly than in younger people. Leask and colleagues (1973) determined blood urea and creatinine levels in fit, elderly subjects who were not taking diuretic therapy. The distribution was log normal and there was a slight increase with age. Men had higher creatinine levels than women, reflecting their greater muscle mass. The reference range for urea using all results combined was 3.9–9.9 mmol/l, and for creatinine was 52–159 µmol/l.

Assessment of renal impairment in the elderly

In younger people, plasma creatinine rather than plasma urea is often used to assess renal function because of the multiplicity of factors which affect the plasma urea level. However, in the absence of renal failure or when renal failure is mild, plasma creatinine is more affected by muscle mass than by renal function. Muscle mass declines in the elderly, so plasma creatinine levels in elderly patients with normal renal function are often remarkably low. Similarly, elderly patients with plasma creatinine levels apparently within the normal range may have significantly impaired creatinine clearance and glomerular filtration rates (Friedman *et al.* 1972, Landahl *et al.* 1981). If creatinine is corrected for body weight, it correlates quite well with glomerular filtration rate. Even so, the correlation between plasma urea and glomerular filtration rate is slightly better (Denham *et al.* 1975). Thus plasma urea is a better guide to renal function in the elderly than is plasma creatinine.

Direct estimation of the glomerular filtration rate, expressed as clearance of endogenous

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creatinine, is difficult in the elderly because of the problems involved in accurate 24-hour urine collections, and is impossible without catheterization if the patient is incontinent. For research purposes, glomerular filtration rate can be estimated using blood samples alone by the radiolabelled chromium-EDTA method (Denham *et al.* 1975, Landahl *et al.* 1981).

Sporn and colleagues (1962) claimed that urinary osmolality was not a useful test in differentiating between reversible oliguria due to dehydration and irreversible oliguria due to established renal failure in the elderly. However, they felt that spot estimation of urinary sodium might be a good test as this was about 6 mmol/l in poor renal perfusion and about 70 mmol/l in established acute tubular necrosis. They studied only 5 cases and their work has yet to be confirmed.

Although, as discussed above, renal function deteriorates with age in the well elderly, this does not apply in ill patients. Here the effects of the illness predominate (Denham *et al.* 1975), and therefore nomograms specifying drug doses in renal failure, which include age as a predictor, may introduce inaccuracy.

The presence of accumulated faeces in the large bowel, so very common in elderly patients, usually makes visualization of the kidneys on a plain abdominal X-ray impossible and preparation for intravenous urography in sick patients is very difficult. Furthermore, intravenous urography is often intolerably uncomfortable for the elderly. It may be difficult to obtain pictures of a satisfactory quality without injecting large amounts of contrast into frail veins. Ultrasonography is therefore the easiest way of imaging the kidneys in elderly patients (Coles *et al.* 1982).

Dehydration in the elderly

Dehydration is the commonest cause of renal impairment (Kafetz & Hodkinson 1982) and frank renal failure (Kumar *et al.* 1973) in elderly patients. By contrast, in patients of all ages, congestive cardiac failure is the commonest cause of renal impairment (Morgan *et al.* 1977). It is not that congestive cardiac failure is any less common in the elderly – indeed, it is as common a cause of uraemia in the elderly as it is at all ages (Kafetz & Hodkinson 1982); rather it is that elderly patients have a particular propensity for dehydration.

There are many possible causes for dehydration in the elderly and a number of different causes operate in each case. There is a concentrating defect within the kidney in the elderly (Mukherjee *et al.* 1973, Rowe *et al.* 1976b). This decline is age-related and independent of the age-related decline in glomerular filtration rate. It is probably caused by a diminution of the renal response to antidiuretic hormone (Miller & Shock 1953). Urinary tract infection worsens this defective concentration (Dontas *et al.* 1968). Thus urinary tract infection is significantly associated with serious dehydration in the elderly (Kafetz & Hodkinson 1982).

It is often said that one of the major factors associated with dehydration in the elderly is a decrease in the thirst drive (Mukherjee *et al.* 1973). However, this is difficult to substantiate. Acute illness in the elderly can often cause confusional states and lack of drinking may be secondary to the confusion rather than caused by a primary abnormality of thirst. The thirst response to fluid loss or deprivation may be mediated by angiotensin and a central dopaminergic mechanism. This can be blocked by haloperidol (Fitzsimons 1976), so patients on this and similar sedatives may truly have a specific abnormality of thirst. Central dopaminergic mechanisms do not seem to be significantly disturbed in Alzheimer-type dementia, although cholinergic mechanisms are markedly impaired (Glen 1980). Acetylcholine is a known intracranial dipsogen (Fitzsimons 1976), so there is a neurochemical basis for supposing that demented patients wish to drink less than they ought.

All the medical conditions that cause dehydration in younger people cause dehydration in the elderly. Inappropriate treatment of the oedema of dependence or inactivity with diuretics is an important iatrogenic cause. Dehydration with hypernatraemia but without hyperglycaemia, well recognized in babies but rare in adult medical practice, is a particular problem in the elderly (Kafetz 1983).

Renal impairment in cardiac failure

Very little work has been done on this common cause of uraemia. The elevation of plasma urea is usually much greater than the elevation of plasma creatinine, suggesting that the uraemia is mainly prerenal (Morgan *et al.* 1977). The commonest explanation for this is that it is due to catabolism of anoxic and congested tissues, particularly muscle tissue (Domenet & Evans 1969, Ujjwal *et al.* 1974). However, the evidence for this is suspect and previous reports of a significant positive correlation between blood urea levels and aspartate transaminase levels have not been confirmed (Kafetz 1982). Furthermore, when a diuresis was produced by frusemide in uraemic elderly patients with cardiac failure, plasma urea fell significantly while plasma creatinine remained the same (Kafetz 1982). This suggested that there was increased urinary excretion during the diuresis because of a tubular rather than a glomerular mechanism. During periods of fluid retention there is increased tubular retention of urea (Goldstein *et al.* 1969) and this is the usual mechanism of prerenal uraemia (Kassirer 1971). Fluid retention with antidiuresis is the predominant activity of the kidney in cardiac failure (Cannon 1977). However, during a diuresis, the distal nephron is relatively impermeable to urea and therefore less is retained (Kassirer 1971). Thus the uraemia of congestive cardiac failure is probably largely due to tubular retention of urea. Diuretic treatment of elderly patients in cardiac failure who also have uraemia generally improves rather than exacerbates the uraemia (Kafetz 1982). However, these patients, as a group, are left with impaired renal function, so there is a clear danger that prolonged diuretic therapy may cause the plasma urea level to rise again.

Other factors in prerenal uraemia

Prerenal factors are involved in the vast majority of cases of renal impairment in the elderly. Apart from those mentioned above, major surgery, use of tetracyclines, hypotension, infection (particularly septicaemia), jaundice and the use of contrast media are involved (Kumar *et al.* 1973).

Intrinsic renal impairment

This is rare but not unknown. The commonest cause is 'pyelonephritis' (Kumar *et al.* 1973). Acute glomerulonephritis is rare but well described (Montoliu *et al.* 1981). Both acute glomerulonephritis with a good prognosis and rapidly progressive glomerulonephritis with a bad prognosis are seen. Acute glomerulonephritis can occur in association with streptococcal skin infections and, if looked for actively, mild episodes may be more common than has been thought. Red blood cell casts have been quite difficult to find in elderly patients with glomerulonephritis, possibly because they are not searched for with sufficient care (Samii *et al.* 1961). Gentamicin therapy is well known to cause renal impairment in the elderly, in particular when some of the factors which cause prerenal uraemia are present. Other, rarer renal diseases which occur are pyonephrosis, hypernephroma (Kafetz & Hodgkinson 1982), acute polyarteritis, focal embolic nephritis and renal vein thrombosis (Kumar *et al.* 1973).

Postrenal renal impairment

By far the commonest cause of postrenal renal impairment in the elderly is prostatic enlargement. In elderly patients with prostatism the glomerular filtration rate is often impaired (Olbrich *et al.* 1957). Transurethral prostatectomy is an operation that lends itself to a variety of different anaesthetic techniques, so there are very few elderly men who are not fit for it. Thus it is well worth looking for urinary obstruction by clinical examination, catheterization to estimate the amount of residual urine and ultrasonography in elderly men with significant renal impairment.

Causes of obstructive renal failure that occur in both sexes are other causes of bladder outflow obstruction, acute fulminating obstructive pyelonephritis, renal stones, carcinoma of the bladder and ureteric obstruction due to carcinomatosis (Kumar *et al.* 1973). In women, obstruction can be caused by fibroids, prolapse and pelvic neoplasm. When obstruction is

relieved by catheterization or prostatectomy, there is often a dramatic diuresis and intravenous fluid replacement is necessary (Thompson 1979). This is due to a mixture of tubular dysfunction (Jamison & Hall 1982) and an osmotic diuresis secondary to uraemia.

Renal replacement therapy in the elderly

If the precipitating factors for acute renal failure are coming under control and the patient has been previously fit, peritoneal or even haemodialysis to allow for renal recovery is perfectly justifiable and successful in the elderly (Kumar *et al.* 1973). There is no indication to support renal function with dialysis when an ill, elderly patient has failure of a number of organs and improvement is not evident. Renal replacement in chronic renal failure is rarely offered to elderly patients in the United Kingdom. However, elsewhere there is a growing experience of this. Haemodialysis can be effective, although there are significant problems in elderly patients which are related to an increased cardiovascular morbidity, vascular access, cardiac failure when there is a large fistula and the use of heparin (Rosen 1976). Cadaveric renal transplantation is probably contraindicated, as the survival rate decreases with age after the age of 50 (Kjellstrand *et al.* 1976).

When renal replacement therapy is being considered in the elderly with chronic renal failure, continuous ambulatory peritoneal dialysis is the treatment of choice. In one series it was found that when comparing elderly patients with younger patients on this treatment, biochemical findings, complications and failure rates were similar in all respects. There were no objective data on quality of life. However, the adjustment of elderly patients to the stresses of this treatment did not appear to differ from that of younger patients, although there was a greater reliance on other family members and on the home dialysis team (Kaye *et al.* 1982).

References

- Cannon P J (1977) *New England Journal of Medicine* **296**, 26–32
- Coles J A, Beynon G P J & Lees W R (1982) *Age and Ageing* **11**, 145–152
- Denham M J, Hodgkinson H M & Fisher M (1975) *Age and Ageing* **4**, 32–36
- Domenet J G & Evans D W (1969) *Quarterly Journal of Medicine* **38**, 117–133
- Dontas A S, Papanayiotou P, Marketos S G & Papanicolaou N T (1968) *Clinical Science* **34**, 73–81
- Fitzsimons J T (1976) *Kidney International* **10**, 3–11
- Friedman S A, Raizner A E, Rosen R, Solomon N A & Sy W (1972) *Annals of Internal Medicine* **76**, 41–45
- Glen I A M (1980) *Hospital Update* **6**, 977–988
- Goldstein M H, Lenz P R & Levitt M S (1969) *Journal of Applied Physiology* **26**, 594–599
- Jamison R L & Hall D A (1982) *Annual Review of Medicine* **33**, 241–254
- Kafetz K (1982) *Journal of Clinical and Experimental Gerontology* **4**, 257–265
- Kafetz K (1983) In: *Clinical Biochemistry in the Elderly*. Ed. H M Hodgkinson. Churchill Livingstone, London & Edinburgh (in press).
- Kafetz K & Hodgkinson H M (1982) *Journal of Clinical and Experimental Gerontology* **4**, 63–70
- Kassirer J P (1971) *New England Journal of Medicine* **285**, 385–389
- Kaye M, Pajel P A & Somerville P J (1982) *Lancet* **ii**, 270–271
- Kjellstrand C M, Shideman J R, Lynch R E, Buselmeier T J, Simmons R L & Najarian J S (1976) *Geriatrics* **31**, 65–73
- Kumar R, Hill C M & McGeowan M G (1973) *Lancet* **i**, 90–91
- Landahl S, Aurell M & Jagenburg R (1981) *Journal of Clinical and Experimental Gerontology* **3**, 29–45
- Leask R G S, Andrews G R & Caird F I (1973) *Age and Ageing* **2**, 14–23
- McLachan M S F (1978) *Lancet* **ii**, 143–146
- Miller J H & Shock N W (1953) *Journal of Gerontology* **8**, 446–450
- Montoliu J, Darnell A, Torras A & Revert L (1981) *Journal of the American Geriatrics Society* **29**, 108–116
- Morgan D B, Carver M E & Payne R B (1977) *British Medical Journal* **ii**, 929–932
- Mukherjee A P, Coni N K & Davison W (1973) *Gerontologia Clinica* **15**, 227–233
- Olbrich O, Woodford-Williams E, Irvine R E & Webster D (1957) *Lancet* **i**, 1322–1324
- Rosen H (1976) *Medical Clinics of North America* **60**, 1105–1119
- Rowe J W, Andres R, Tobin J D, Norris A H & Shock N W (1976a) *Journal of Gerontology* **31**, 155–163
- Rowe J W, Shock N W & DeFronzo R A (1976b) *Nephron* **17**, 270–278
- Samiy A H, Field R A & Merrill J P (1961) *Annals of Internal Medicine* **54**, 603–609
- Sporn I N, Lamestremere R G & Papper S (1962) *New England Journal of Medicine* **267**, 130–132
- Thompson F D (1979) *British Journal of Hospital Medicine* **21**, 46–56
- Ujjwal J S, Bhatnagar H N S & Bordia A (1974) *Journal of the Association of Physicians of India* **22**, 589–596